

ORIGINAL ARTICLE

Size for gestational age at birth: impact on risk for sudden infant death and other causes of death, USA 2002

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Background: Small for gestational age (SGA) infants have been reported to be at higher risk for sudden infant death syndrome (SIDS).

Objective: To compare the risk of SIDS among SGA and large for gestational age (LGA) infants with that of death from other causes of sudden unexpected deaths in infancy (SUDI) and the residual "other" causes of infant death.

Methods: The 2002 US period infant birth and death certificate linked file was used to identify infant deaths classified as SIDS (ICD-10 code R95), SUDI (ICD-10 codes R00–Y84 excluding R95) or all other residual codes. The 2002 race and sex-specific birth cohorts were used to generate the 10th and 90th percentiles of birth weight for each gestational age week from 24 to 42 weeks' gestation. Demographic variables previously identified as associated with SIDS were used in multiple logistic regression equations to determine the risk for death among SGA and LGA infants (birth weight <10th percentile and >90th percentile, respectively) independent of other potentially confounding variables.

Results: Complete data on 1956 SIDS deaths, 2012 SUDI, and 11 592 other deaths were available. The adjusted OR for SIDS, SUDI and "other" causes for SGA infants was 1.65 (95% CI 1.47 to 1.85), 1.78 (1.59 to 2.00) and 4.68 (4.49 to 4.88), respectively. The adjusted OR for LGA infants was reduced for SIDS (0.73 (0.60 to 0.89)), SUDI (0.81 (0.68 to 0.98)) and "other" (0.42 (0.38 to 0.46)).

Conclusion: Although SGA infants seem to be at slightly increased risk for SIDS or SUDI their risk for "other" residual causes is about 2.5 times higher. LGA infants seem to be at reduced risk of mortality for all causes. The mechanisms by which restricted intrauterine growth increases risk of mortality and excessive intrauterine growth offers protective effects are uncertain.

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Small for gestational age (SGA) infants have been reported to be at 1.4 to 2.0 times greater risk for sudden infant death syndrome (SIDS).^{1–5} Other causes of death have also been reported to be associated with being SGA by some authors,^{6–7} in particular, preterm SGA infants are reported to have an increased risk of death ranging from 2.4 to 3.6 times that of appropriate for gestational age (AGA) preterm infants.^{8–9} Other authors, however, have observed no increase in risk for other causes of death among term SGA infants after adjusting for racial disparities in birth weight.¹⁰ The mechanisms associated with the increased risk for SIDS among SGA infants remain unclear, but some investigators have suggested that the risk may be secondary to the hypoxia these infants are suspected of being exposed to in utero.^{11–12} Why in utero hypoxia may make an infant more vulnerable to SIDS has been hypothesised to be related to a reduction of serotonergic receptors in multiple brainstem nuclei.^{13–14}

Despite the biological plausibility of the hypoxic-related vulnerability of SGA infants for SIDS, comparative studies of just how much greater risk such an infant has for SIDS compared with other causes of death are not replete in the literature. Thus, the purpose of this analysis was to determine whether SGA infants were at greater risk for SIDS compared with other causes of death. The risk of large for gestational age (LGA) infants for SIDS has not been closely examined and an examination of this relationship was thus included in the analysis. Because of a trend towards the reclassification of SIDS deaths to other causes of sudden unexpected causes of death (SUDI),¹⁵ the risk of SUDI excluding SIDS for SGA infants as well as the risk of the remaining residual "other" causes of death among these infants was examined for the USA for the year 2002.

METHODS

The 2002 US period infant birth and death certificate linked vital statistics file was used to identify survivors, infant deaths from day 1 to 354 due to either SIDS (International Classification of Diseases (ICD)-10 code R95) or SUDI (ICD-10 codes R00–Y84 excluding R95) or all "other" residual codes.^{16–17} Demographic characteristics previously identified as associated with SIDS¹⁸ and available from the linked vital statistics files were maternal race, maternal place of birth, maternal education, birth number, maternal tobacco usage, presence of maternal diabetes, pregnancy-induced hypertension, pregnancy weight gain, sex of infant, birth weight of infant and infant gestational age. The 10th and 90th percentile values for birth weight for each week of gestational age from 24 to 42 weeks for each sex within each race-specific category using the 2002 birth cohort was obtained using SAS Proc Univariate.¹⁹ Infants with birth weights less than the 10th percentile for their gestational age, sex and race were labelled SGA, those between the 10th and 90th percentiles as AGA and those greater than the 90th percentile as LGA. Analysis was limited to infants with birth weights of 500–6000 g.

Frequency distributions of the various covariates were determined using SAS Proc Frequency and significant differences in the distributions determined using Mantel–Haenszel χ^2 values. Logistic regression models were used to determine the risk of death among infants who were small and large for gestational age independent of potential confounding variables and for examining significant interactions between variables

Abbreviations: AGA, appropriate for gestational age; LGA, large for gestational age; SGA, small for gestational age; SIDS, sudden infant death syndrome; SUDI, sudden unexpected death in infancy

using SAS Proc Logistic. An arbitrary p value of <0.05 was used to indicate statistical significance. Because SUDI “unexplained” deaths differed significantly from SUDI “explained” deaths only on prevalence of multiple births (5.0% v 3.3%, respectively) and maternal education (40.8% <12 years v 38.7%, respectively), and did not differ in the prevalence of small and large for gestational age infants, the two categories were combined and are presented as total SUDI for the logistic regression analyses.

RESULTS

There were 3 273 544 survivors, 1956 SIDS deaths, 2012 SUDI deaths and 11 592 “other” deaths with all variables present. The most prevalent cause of SUDI was “other ill-defined/unspecified causes” (ICD-10 code R99) designated as “unexplained” SUDI and comprised 40.4% of all SUDI deaths. “Accidental suffocation in bed” (ICD-10 code W75) representing 18.7% of the total number of SUDI deaths, comprised the largest proportion of “explained” SUDI (table 1). The most prevalent “other” causes were “congenital malformation of the heart, unspecified” (ICD-10 code Q249) and “extreme immaturity” (ICD-10 code P072), representing 4.5% and 4.0% of the total “other” cause deaths, respectively (table 1).

The mean birth weight for survivors was 3299 g compared with 2959 g for SIDS victims (p<0.05), 2911 g for SUDI-unexplained victims (p<0.05), 2994 g for SUDI-explained and 1919 g (p<0.05) for “other” causes of death. The mean gestational age for survivors was 38.5 weeks compared with 37.6 weeks for SIDS victims (p<0.05); 37.4 weeks for SUDI-unexplained victims (p<0.05), 37.7 weeks for SUDI-explained (p<0.05) and 32.7 weeks for “other” causes (p<0.05).

Non-Hispanic black infants were over-represented among all causes of death compared with survivors (15.5%). Among SIDS victims, 28.5% were non-Hispanic black, and among SUDI-unexplained 32.2%, among SUDI-explained 34.0% and among “other” causes 25.4% were non-Hispanic black. Among SIDS and SUDI deaths mothers were more likely to: be born within

the USA than among those infants who died of “other” causes; have fewer than 12 years of education; and use tobacco during pregnancy. Also, the infant’s sex among SIDS and SUDI deaths was more likely to be male. Multiple births were more frequent among infants dying of all causes compared with survivors and most prevalent among infants dying of “other” causes (13.3%) than among SIDS (5.8%). There was a higher prevalence of maternal diabetes and pregnancy-induced hypertension among infants dying of “other” causes. Pregnancy weight gain was similar among survivors, and SIDS and SUDI victims, but it was reduced among victims of “other” causes. SGA infants were more prevalent among SIDS (18.8%), SUDI-unexplained (19.7%), and SUDI-explained (18.4%) deaths compared with survivors (9.7%). Nevertheless, the prevalence of SUDI-unexplained infants was highest among infants dying of “other” causes (31.8%, p<0.01). There were fewer LGA infants among all categories of death when compared with survivors. The mean age at death was greatest for SUDI-explained causes and least for “other” causes.

Logistic regression was used to model the risk for the various causes of death among infants who were small compared with infants who were appropriate for gestational age. The unadjusted odds ratio for SIDS versus survivors among small compared with appropriate for gestational age infants was 2.03 (95% CI 1.82 to 2.27), and for SUDI it was 2.08 (1.87 to 2.32) and for “other” causes 4.19 (4.04 to 4.35). Following adjustment for potentially confounding variables, the adjusted odds ratio for SIDS among small compared with appropriate for gestational age infants was 1.65 (1.47 to 1.85), and for SUDI it was 1.78 (1.59 to 2.00) and for “other” causes 4.68 (4.49 to 4.88) (table 3). LGA infants were at reduced risk for all causes of death (table 3).

Significant interactions were observed between size and gestational age for SUDI and “other” causes, and between size and tobacco for SUDI and “other” causes. Logistic models were developed to determine the risk of SIDS, SUDI and “other” causes for SGA infants stratified by gestational age (table 4). Compared with infants who were appropriate for gestational age those who were small were at greater risk for SIDS across all gestational age categories. Only the more mature SGA infants (>33 weeks) were at greater risk for SUDI. The least mature SGA infants (24–32 weeks) were at an increased risk for “other” causes (adjusted odds ratio 3.92), but those at >33 weeks were at even greater risk (adjusted odds ratio 5.96 and 5.21 for 33–36 weeks and 37–42 weeks, respectively). In contrast, LGA infants were at reduced risk for all causes of death across all gestational age categories.

Logistic models stratified by size and gestational age showed a consistent increased risk for SIDS and SUDI among all sizes of infants of women who smoked compared with non-smokers in all gestational age categories (table 5). The number of infants born to smokers for infants small and large for gestational age at gestational ages of 24–32 weeks and whose death was categorised SIDS or SUDI was very small and does not provide reliable estimates. There was no increased risk of “other” causes of death among infants who were small or large for gestational age whose mother smoked in any of the gestational age categories except among AGA term infants, where there was a slight increased risk (odds ratio 1.18). For most gestational age and size categories there was at least a trend to a reduced risk of “other” causes of death among smoking mothers.

DISCUSSION

The present analysis confirms other reports that have identified an increased risk of SIDS among SGA infants.^{1–5 8 9} Because of the observation of a shift in the classification of SIDS deaths,

Table 1 Most prevalent causes among sudden unexpected deaths in infancy and “other” categories

Cause	ICD-10 code	No. of deaths	Per cent of total deaths in category
Sudden unexpected deaths in infancy (N=2012)			
Unexplained			
Other ill-defined and unspecified causes	R99	812	40.4
Explained			
Accidental suffocation in bed	W75	377	18.7
Assault	Y09	101	5.0
Unspecified threat to breathing	W84	83	4.1
Maltreatment	Y079	60	3.0
Suffocation (undetermined intent)	Y20	47	2.3
Other threats to breathing	W83	41	2.0
Exposure to fire	X00	32	1.6
Drowning in bathtub	W65	28	1.4
Motor vehicle accident	V892	27	1.4
“Other” residual causes of death (N= 11 592)			
Congenital malformation of heart (unspecified)	Q249	522	4.5
Respiratory distress syndrome	P220	465	4.0
Extreme immaturity (<28 weeks’ gestation)	P072	451	3.9
Pulmonary hypoplasia	Q336	389	3.4
Trisomy 18	Q913	370	3.2
Bacterial sepsis (unspecified)	P369	369	3.2
Birth asphyxia	P219	300	2.6
Necrotising enterocolitis	P77	269	2.3
Hypoplastic left heart	Q234	261	2.3
Trisomy 13	Q917	219	1.9

Table 2 Per cent distribution of selected population characteristics by survival status, USA, 2002

Characteristic	Survivors N = 3 271 532	SIDS N = 1956	SUDI		"Other" causes N = 11 592
			Unexplained N = 812	Explained N = 1200	
Race					
Hispanic					
Cuban	0.4	0.0	0.1	0.1	0.3
Latin	2.9	1.1	1.1	1.3	2.5
Mexican	11.3	6.7	8.1	8.2	10.3
Puerto Rican	1.6	1.6	1.6	1.3	1.8
Non-Hispanic black	15.5	28.5	32.2	34.4	25.4
Non-Hispanic white	61.5	56.5	52.2	49.6	54.1
Other	6.8	5.7	4.8	5.3	5.6
p Value for general association		<0.01*	<0.01*	<0.01*	<0.01*
			0.02†	<0.01†	<0.01†
				NS‡	
Mother's place of birth					
USA	80.3	93.1	91.5	91.8	83.2
Outside USA	19.5	6.8	8.1	8.0	16.4
Unknown	0.2	0.1	0.4	0.2	0.4
p Value for general association		<0.01*	<0.01*	<0.01*	<0.01*
			0.03†	0.04†	<0.01†
				NS‡	
Multiple birth					
Yes	3.3	5.8	5.1	3.2	13.3
p Value for general association		<0.01*	0.05*	NS*	<0.01*
			0.02†	<0.01†	<0.01†
				0.05‡	
Maternal education					
<12 years	21.0	38.5	43.2	39.1	25.8
12 years	31.4	39.5	36.6	40.6	35.6
13–16 years	38.1	19.6	18.2	19.0	31.8
>16 years	10.4	2.4	2.0	1.3	6.8
p Value for general association		<0.01*	<0.01*	<0.01*	<0.01*
			0.02†	0.05†	<0.01†
				0.03‡	
Maternal diabetes	3.4	2.8	3.2	3.3	4.0
p Value for general association		NS*	NS*	NS*	<0.01*
			NS†	NS†	<0.01†
				NS‡	
Pregnancy-induced hypertension	4.1	4.2	3.8	4.2	4.8
p Value for general association		NS*	NS*	NS*	<0.01*
			NS†	NS†	NS†
				NS‡	
Pregnancy weight gain (lb), mean (SD)	34.5 (20.4)	34.7 (24.1)	34.9 (24.3)	34.8 (23.2)	32.9 (27.2)
p Value for Bonferroni t tests		NS*	NS*	NS*	<0.05*
			NS†	NS†	<0.05†
				NS‡	
Tobacco use					
Yes	11.3	36.5	34.1	32.5	14.8
p Value for general association		<0.01*	<0.01*	<0.01*	<0.01*
			NS†	0.02†	<0.01†
				NS‡	
Sex					
Male	51.2	59.4	60.0	56.4	56.1
Female	48.8	40.6	40.0	43.6	43.9
p Value for general association		<0.01*	<0.01*	<0.01*	<0.01*
			NS†	NS†	<0.01†
				NS‡	
Gestational age (weeks)					
24–32	2.3	6.9	7.6	7.2	46.0
33–36	10.0	17.5	21.4	16.5	17.4
37–42	87.7	75.6	71.0	76.3	36.6
p Value for general association		<0.01*	<0.01*	<0.01*	<0.01*
			NS†	NS†	<0.01†
				0.04‡	
Birth weight (g)					
500–1499	1.1	4.1	4.0	4.8	43.4
1500–2499	6.6	16.6	20.3	15.0	21.9
2500–6000	92.3	79.3	75.7	80.2	34.7
p Value for general association		<0.01*	<0.01*	<0.01*	<0.01*
			NS†	NS†	<0.01†
				0.01‡	

Table 2 Continued

Characteristic	Survivors N = 3 271 532	SUDI			"Other" causes N = 11 592
		SIDS N = 1956	Unexplained N = 812	Explained N = 1200	
Size for gestational age					
Small (<10 percentile)	9.7	18.8	19.7	18.4	31.8
Large (>90th percentile)	9.7	5.7	6.2	6.8	3.5
Appropriate	80.6	75.5	74.1	74.8	64.7
p Value for general association		<0.01*	<0.01*	<0.01*	<0.01*
			NS†	NS†	<0.01†
Age at death (days), mean (SD)		95 (61)	92 (74)	12 (94)	44 (74)
p Value for Bonferroni t tests			NS†	0.05†	0.05†
				0.05‡	

NS, non-significant; SIDS, sudden infant death syndrome; SUDI, sudden unexpected death in infancy.

*p Value determined for distributions compared to survivors.

†p Value determined for distributions compared with SIDS.

‡p Value determined comparing SUDI-unexplained to SUDI-explained.

we also examined the prevalence of SGA infants among infants whose death was categorised SUDI.¹⁵ SGA infants were equally prevalent among SUDI victims, for the most part consisting of infants labelled as dying with "ill-defined and unspecified conditions" and "suffocation". The implication that the risk of SIDS and SUDI is comparable among SGA infants suggests that there is a great deal of cross-classification of the cause of death between SIDS and SUDI. Alternatively, it suggests a mechanism by which being SGA affects a greater vulnerability to death that is similar in both SIDS and SUDI classified deaths. However, the biological plausibility of a similar mechanism by which being SGA increases vulnerability for SIDS and SUDI seems a little difficult to reconcile because of the externally imposed nature of the causes of most SUDI, such as suffocation. The SGA infant's vulnerability for SIDS has been hypothesised to be related to hypoxic in-utero events that may affect arousability.¹¹⁻²⁰ Thus, it seems that either cross-classification issues or potentially two different mechanisms could be working to make the SGA infant more vulnerable to SIDS and SUDI. Nevertheless, the reduction in the odds ratio from 2.0 to 1.6 for SIDS and from 2.1 to 1.7 for SUDI among SGA infants when potentially confounding sociodemographic variables are controlled in the estimation of the odds ratio suggests that a fair amount of unadjusted risk is accounted for by environmental and behavioural factors for both classifications. Although it is undeniable that some increased risk of SIDS or SUDI exists among SGA infants, the risk for "other", in

particular, perinatal causes of mortality, among SGA infants is much greater (odds ratio 4.15 and adjusted odds ratio 4.68).

Is being SGA an indicator of enhanced vulnerability to SIDS and should it be construed as being in the causal pathway of death from SIDS? As the risk of being SGA among SIDS victims was reduced with socioeconomic adjustment, being an SGA SIDS victim may be merely a proxy for environmental and behavioural conditions that lead to intrauterine growth restriction, rather than being SGA making the infant biologically vulnerable to SIDS or SUDI. Over and above the impact of intrauterine growth restriction, we found maternal tobacco usage to be an independent risk factor for SIDS and SUDI, but not for "other" causes of death. Other investigators have reported the consistent risk of SIDS associated with tobacco usage independent of the intrauterine growth restriction effect associated with tobacco use.²¹⁻²³ The observation among infants who were appropriate or large for gestational age that tobacco use is also associated with increased risk of dying of SIDS and SUDI suggests a mechanism for tobacco's purveyance of that increased risk that is not associated with tobacco's growth restricting effects. Tobacco use was not associated with an increased risk for "other" causes of death. Thus, it is possible that tobacco use may be a proxy for maternal risk-taking behaviour rather than tobacco directly affecting the biological vulnerability of the infant. In contrast, the protective effect of growth acceleration in utero has received little attention. That the risks for all causes of death were reduced among LGA

Table 3 Adjusted odds ratios* (95% CI) for mortality from specified category of death by size at birth

Size for gestational age	Specific category v survivors		
	SIDS (n = 1956)	SUDI (n = 2012)	"Other" causes (n = 11 592)
Small (<10th percentile)	1.65 (1.47 to 1.85)	1.78 (1.59 to 2.00)	4.68 (4.49 to 4.88)
Large (>90th percentile)	0.73 (0.60 to 0.89)	0.81 (0.68 to 0.98)	0.42 (0.38 to 0.46)
Appropriate	1.00 (reference)	1.00 (reference)	1.00 (reference)
Size for gestational age	Specific category v SIDS		
	SUDI		"Other" causes
Small (<10th percentile)	1.08 (0.92 to 1.27)		2.66 (2.33 to 3.04)
Large (>90th percentile)	1.13 (0.87 to 1.47)		0.88 (0.69 to 1.11)
Appropriate	1.00 (reference)		1.00 (reference)

*Odds ratios adjusted for maternal race, mother's place of birth, maternal education, multiple birth, tobacco use, maternal diabetes, presence of pregnancy-induced hypertension, pregnancy weight gain, gestational age, and infant sex.

Table 4 Adjusted odds ratio* (95% CI) for specified mortality category of death stratified by size and gestational age

Gestational age (weeks)	Size for gestational age		
	Appropriate	Small	Large
Sudden infant death syndrome			
24–34	1.00 (reference), 106†	1.88 (1.13 to 3.11), 18	0.78 (0.43 to 1.42), 12
33–36	1.00 (reference), 262	1.57 (1.17 to 2.11), 57	0.77 (0.50 to 1.17), 23
37–42	1.00 (reference), 1108	1.64 (1.44 to 1.88), 293	0.69 (0.55 to 0.88), 77
Sudden unexpected death in infancy			
24–32	1.00 (reference), 119	1.24 (0.71 to 2.17), 14	0.89 (0.53 to 1.48), 15
33–36	1.00 (reference), 286	1.56 (1.16 to 2.09), 54	0.95 (0.66 to 1.38), 32
37–42	1.00 (reference), 1094	1.85 (1.62 to 2.10), 313	0.77 (0.62 to 0.96), 85
"Other" residual causes of death			
24–32	1.00 (reference), 3844	3.92 (3.66 to 4.19), 1414	0.15 (0.12 to 0.19), 80
33–36	1.00 (reference), 1197	5.96 (5.41 to 6.56), 718	0.58 (0.47 to 0.71), 96
37–42	1.00 (reference), 2458	5.21 (4.88 to 5.57), 1557	0.77 (0.67 to 0.88), 281

*Odds ratios adjusted for maternal race, maternal place of birth, multiple birth, maternal education, maternal tobacco use and infant sex.

†Number of deaths within specific size and gestational age strata.

Table 5 Adjusted odds ratios* (95% CI) for specified categories of mortality for infants whose mothers used tobacco and non-tobacco users stratified by size and gestational age

Gestational age (weeks)	Size for gestational age		
	Appropriate	Small	Large
Sudden infant death syndrome			
24–34	2.15 (1.41 to 3.28), 40†	1.48 (0.18 to 2.50), 3	3.02 (0.82 to 10.9), 4
33–36	2.79 (2.12 to 3.68), 94	2.33 (1.31 to 4.15), 25	2.25 (0.84 to 6.06), 6
37–42	2.98 (2.60 to 3.42), 375	2.66 (2.06 to 3.45), 148	3.85 (2.20 to 6.71), 18
Sudden unexpected death in infancy			
24–32	1.42 (0.91 to 2.20), 31	2.34 (0.65 to 8.33), 4	4.63 (1.56 to 13.9), 7
33–36	2.74 (2.11 to 3.57), 108	1.15 (0.62 to 2.13), 17	2.97 (1.30 to 6.76), 9
37–42	2.62 (2.27 to 3.01), 332	2.63 (2.05 to 3.38), 152	1.06 (0.48 to 2.34), 7
"Other" residual causes of death			
24–32	0.87 (0.79 to 0.96), 555	1.05 (0.88 to 1.24), 227	0.97 (0.49 to 1.90), 11
33–36	1.02 (0.87 to 1.21), 181	0.67 (0.54 to 0.84), 112	0.80 (0.41 to 1.58), 10
37–42	1.18 (1.04 to 1.32), 340	0.74 (0.64 to 0.85), 269	0.99 (0.56 to 1.76), 13

*Odds ratios adjusted for maternal race, maternal place of birth, multiple birth, maternal education and infant sex.

†Number of deaths in which mother used tobacco within specific size and gestational age strata.

infants begs the question of how growth acceleration might be beneficial. This observation is reported after controlling for potentially confounding conditions such as the presence of maternal diabetes and pregnancy weight gain. Whether accelerated in-utero growth might reflect greater maturation of other organ systems than simply an increase in fetal mass deserves further attention.

Our analysis is subject to the limitations of all analyses that use vital statistics data. The best estimate gestational age computed by a National Center for Health Statistics algorithm offers a reasonable estimate but does not guarantee the best estimate which would entail ultrasound dating of pregnancies. Maternal variables such as age, race, education and tobacco use are self-reported and subject to recall bias. Nevertheless, the large number of deaths available for analysis in this dataset makes it an important source for the production of the most reliable estimates of national risk. Another major limitation of vital statistics data in the USA is the lack of precision in certifying the type of sudden unexpected infant death category in which an infant is placed. Compared with case-control studies with death scene investigations and parental interviews that allow greater precision in determining the "unexplained" or "explained" nature of a sudden unexpected death, no such process is standardised in the designation of a category of death in the vital statistics data for the USA.^{15 24 25} Thus the great risk for cross-classification of deaths ascribed to SIDS and SUDI in this analysis.

Poverty, environmental conditions, maternal behaviour that includes exposure to tobacco products, and low educational attainment are greater risk factors for SIDS than is fetal growth

What is already known on this topic

- Small for gestational age infants are at greater risk for sudden infant death syndrome.
- A number of risk factors, such as tobacco usage, pregnancy-induced hypertension and pregnancy weight gain, are associated with intrauterine growth restriction.

What this study adds

- Although small for gestational age infants are at slightly higher risk for sudden infant death, these infants are at greater risk for "other" causes.
- Growth acceleration appears protective for sudden infant death syndrome as well as "other" causes independent of maternal diabetes and pregnancy weight gain.

restriction.^{26, 27} That the alteration of infant care practices and the immediate environment of an infant can affect SIDS mortality is now well documented by the "Back to Sleep" programme.²⁸ Greater attention towards better parenting education and infant safe environments may provide more rapidly for further reductions in SIDS and SUDI mortality while our understanding of the biological basis for vulnerability to these causes of mortality moves more slowly forwards.

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